than 800 ml) or a decreased peak flow rate (less than 60 liters per minute) are laboratory findings that indicate a severe attack. All of these factors should be assessed while a patient is receiving maximal bronchodilator therapy. If a patient fails to respond, intravenous corticosteroid therapy should be instituted promptly, with the recognition that the onset of effect on the asthmatic airway following intravenous administration of a corticosteroid has been determined to be two hours.[24,25] A delay in the use of the drug deprives a patient of its potential benefits. Corticosteroids should also be administered to patients with severe asthmatic exacerbations who have received systemic corticosteroids in the previous 12 months.

The development of infections in patients immunosuppressed by corticosteroids is well documented. Although infrequent, fatal infections can occur in asthmatic patients receiving corticosteroids. Patients must be carefully monitored for infections and other complications. Prompt treatment of complications helps avert fatal outcomes.

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Chest Wall Stimulation for Pacemaker Inhibition

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PROPER SENSING FUNCTION of a ventricular-inhibited (demand) pacemaker generator requires a stable intracardiac signal having sufficient amplitude, duration and slew (rate of change in voltage).[1-3] Intracardiac signals that do not meet the sensing requirements of the pacemaker generator may be sensed intermittently or not at all, resulting in the delivery of inappropriately early pacing stimuli. If these stimuli fall during a critical time period during ventricular repolarization, repetitive ventricular beating may result. Chest wall stimulation, during which electrical stimuli are delivered to the body surface via an external pacemaker generator, can produce signals capable of being sensed by the implanted demand pacemaker generator, which will then inhibit.[4]

We report the case of a patient with acute myocardial infarction in whom chest wall stimulation was used successfully for 12 hours in order to inhibit an implanted pulse generator that caused repeated episodes of ventricular tachycardia and fibrillation because of failing to sense intracardiac signals.

Report of a Case

A 77-year-old man was admitted to the coronary care unit complaining of chest pain unrelieved by sublingual nitroglycerin. Three years earlier he had had a nontransmural anterior myocardial infarction complicated by bradycardia-tachycardia syndrome and bundle branch block, and a permanent unipolar ventricular demand pacing system (Ela Stilith 60) was implanted and its rate set at 51 per minute. Because of stable postinfarction angina, shortness of breath and periodic supraventricular tachycardias, the patient was placed on a daily regimen of 0.25 mg of digoxin, 1.4 grams of quinidine gluconate, 80 mg of propranolol and sublingual nitroglycerin tablets.

On physical examination at admission the patient appeared to be in respiratory distress, coughing blood-tinged sputum. Blood pressure was 150/110 mm of mercury and apical pulse 100 per minute and irregular. Pertinent findings included normal central venous pressure, bilateral rales, a sustained nondisplaced left ventricular apical impulse and an S_3 gallop. Results of the following laboratory studies were within normal limits: complete blood count, analysis of urine, blood urea nitrogen level and electrolyte determinations, including serum potassium. Arterial blood gas analysis showed

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pH of 7.2; P_{CO_2} was 38 and P_{O_2} 51 mm of mercury. An x-ray study of the chest showed cardiomegaly and pulmonary edema. An electrocardiogram showed atrial fibrillation, rapid ventricular rate, right bundle branch block pattern and acute transmural anterior wall myocardial infarction. Pacemaker sensing and pacing functions were normal.

The patient was treated with oxygen, morphine sulfate and intravenously given furosemide with some resolution of his pulmonary edema. Serial electrocardiograms and creatine kinase determinations documented acute myocardial infarction. The quinidine gluconate and propranolol administration was discontinued. The patient's course over the ensuing 24 hours was complicated by low cardiac output, congestive heart failure, intermittent atrial fibrillation with rapid ventricular rate (requiring additional doses of intravenous digoxin to a serum level of 2.9 μg per ml to lower ventricular rate) and episodes of ventricular tachycardia, flutter and fibrillation for which he received lidocaine intravenously. Many of these ventricular arrhythmias, as well as normal QRS complexes, were not sensed by the pacemaker, which continued to deliver pacing stimuli (Figures 1 through 3). On two separate occasions pacemaker-induced ventricular flutter and fibrillation resulted from unsensed spontaneous QRS complexes (Figure 4); these episodes were successfully treated with electrical defibrillation.

Because of the life-threatening nature of the pacemaker-induced ventricular arrhythmias, the implanted pulse generator was inhibited using the technique of chest wall stimulation. Suction cup electrodes were placed on the skin as follows: one at the fourth left interspace at the left sternal border, where the intracardiac (cathodal) electrode was considered to be located; and the other over the implanted pacemaker generator, which served as the anode in this unipolar pacing system. The skin electrodes were then connected to an external pacemaker generator (Medtronic Model 5880) via cables whose ends are specially designed to fit into the terminals of the external pulse generator. The rate of the external generator was set to 70 per minute in order to override the rate of the implanted generator, and the mode of function set to asynchronous (fixed rate). The external pacemaker generator was then turned on and its output gradually increased. At about 3 mA the chest wall stimuli were of sufficient amplitude to be sensed by the implanted pulse generator, which was consequently inhibited.

Chest wall stimulation was continued over the next 12 hours, without patient discomfort and without further pacer-induced arrhythmias. Despite aggressive

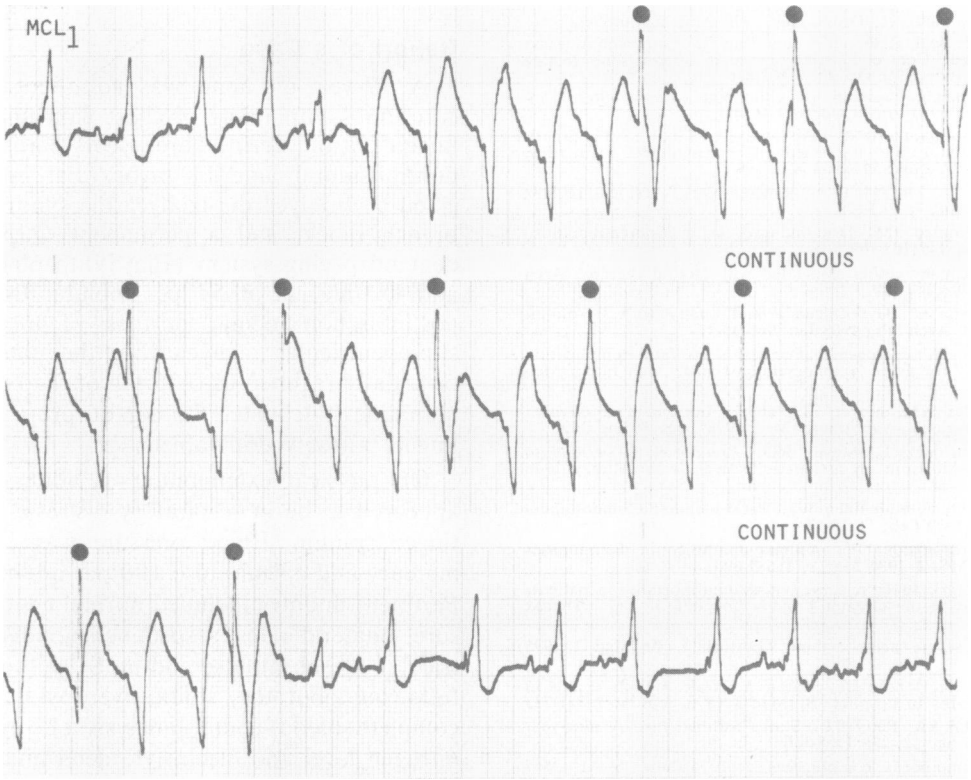

Figure 1.—These three continuous MCL_1 rhythm strips show sinus rhythm with right bundle branch block, a fusion complex (fifth QRS complex) and ventricular tachycardia. The ventricular tachycardia is initially sensed by the pacemaker generator as evidenced by the absence of pacing artifacts. Toward the end of the top strip, however, pacing artifacts occur, and continue at regular intervals corresponding to a rate of about 50 per minute **(closed circles).** Ventricular capture by the pacemaker occurs (third and eighth QRS complexes in the middle strip and fifth QRS complex in the bottom strip), and may actually have fortuitously resulted in tachycardia termination.

medical management, however, the patient died on the third hospital day.

Discussion

The occurrence of acute myocardial infarction provides a potential milieu for pacemaker-related problems due both to undersensing and to changing myocardial stimulation thresholds, as well as changing thresholds for the development and maintenance of repetitive ventricular arrhythmias, including ventricular fibrillation.

Sensing problems in this patient could have been due to electrode catheter displacement, pulse generator malfunction or undersensing resulting from intracardiac signals of too low a voltage, too slow a rate of change of voltage (slew) or prolonged duration (or both of the latter). Pulse generator malfunction was unlikely, as the lithium-powered unit had been in place for three years; in addition, normal pacing function was present, and normal sensing capability was shown by its inhibition by chest wall stimuli. Electrode catheter displacement, albeit possible, was also extremely unlikely because (1) the lead had been in place for three years, (2) *ventricular* pacing was occurring, (3) x-ray films of the chest did not suggest major changes in position and (4) both the pacing artifact axis and contour and axis

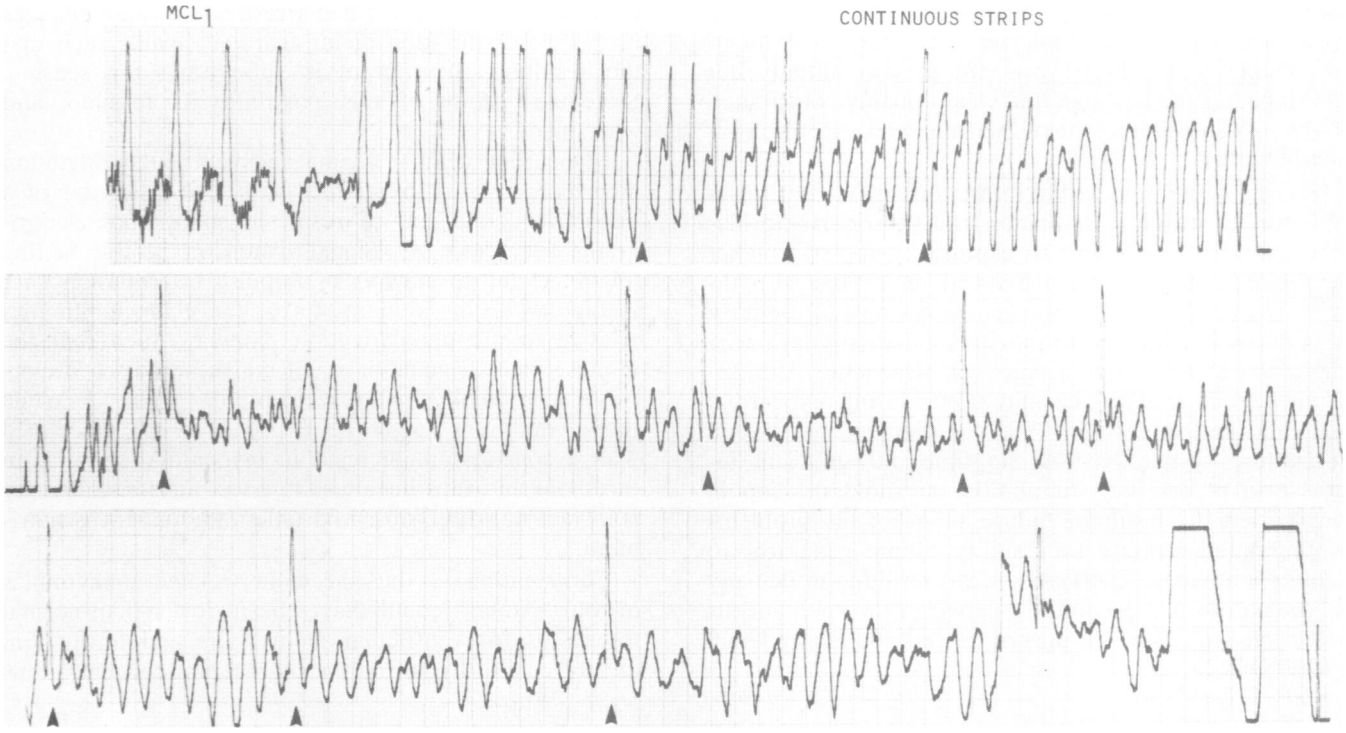

Figure 2.—These continuously recorded MCL_1 rhythm strips show atrial flutter with rapid (2:1 and 3:1) ventricular response. Polymorphous ventricular tachycardia degenerating to ventricular fibrillation occurs abruptly. The pacemaker does not sense the tachycardia and fibrillation, as indicated by the delivery of pacing artifacts **(arrows).** However, since the interval between pacing artifacts is not regular at 1,200 ms (50 beats per minute), it may be assumed that intermittent sensing of ventricular electrical activity is occurring. In the middle strip two pacing artifacts occur at an interval of about 600 ms (100 beats per minute). As the magnetic rate of this pacemaker generator is the same as the automatic (pacing) rate, conversion to fixed rate mode in the presence of electrical "noise" caused by the ventricular tachycardia cannot explain the observation.

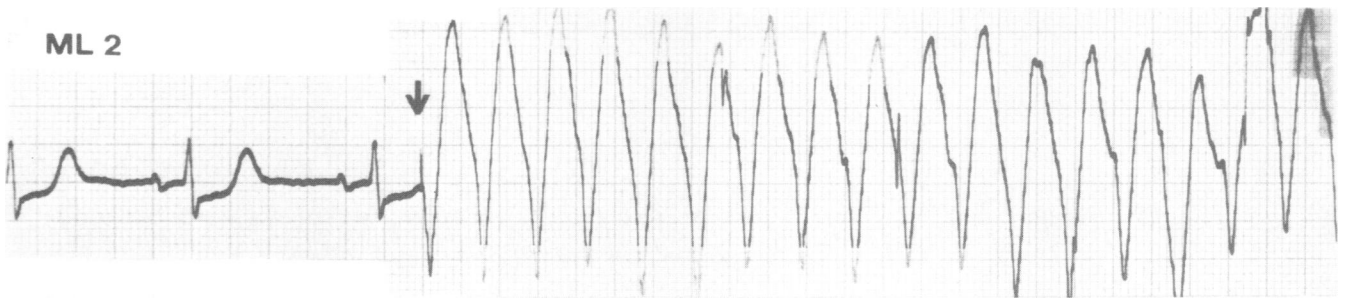

Figure 3.—Failure to sense a sinus-initiated QRS complex results in the delivery of a pacing stimulus at the onset of the inscription of the T wave, with consequent development of ventricular tachycardia. Partial sensing of this tachycardia is occurring, as evidenced by the intermittency of the pacing artifacts. Note that the unsensed QRS complex appears on the surface electrocardiogram to be no different from other, sensed, QRS complexes, illustrating that the surface electrocardiogram does not reflect the quality of intracardiac signals.

of paced QRS complexes were identical to those recorded at the time of pacing system implant.

The most likely cause of sensing problems in this patient lay in the suboptimal nature of the intracardiac signal. Signals of borderline voltage, long duration or poor intrinsic deflection may fall below the sensing threshold of a permanent pacemaker.[1,3] Such signals may occur in the setting of conduction system disease,[5] during acute myocardial infarction,[6,7] when impulses originate in ventricular tissue, when abnormalities of pH or electrolytes—especially potassium—are present, and when high concentration of type I antiarrhythmic agents (quinidine, procainamide, disopyramide) exist. As several of these conditions may coexist in very ill patients, it is not always possible to define a precise cause of pacemaker undersensing. In our patient, pacemaker undersensing was not present initially but developed in the hospital, and was probably contributed to by a deteriorating hemodynamic and electrophysiologic status.

During acute myocardial infarction, changing thresholds for myocardial stimulation and ventricular fibrillation can result in failure to capture by the pacemaker or proclivity to pacemaker-induced ventricular arrhythmias (or both). While pulse generators now in use have energy outputs too low to precipitate arrhythmias under the *usual* clinical circumstances in which they are implanted, such arrhythmias may occur during myocardial ischemia and infarction,[8-11] hyperadrenergicity[12,13] and premature systoles.[14-16] At no time did sufficient increase in myocardial stimulation threshold develop in our patient to result in failure to pace; his problems were related entirely to inability of the generator to sense spontaneous QRS complexes, resulting in delivery of premature pacing stimuli relative to the spontaneous RR cycle length, which in turn caused potentially lethal arrhythmias.

The changing quality of intracardiac signals that causes intermittent nonsensing of spontaneous QRS complexes might be related to (1) ischemia and necrosis, (2) metabolic acidosis, (3) ventricular dilatation, (4) myocardial cellular edema, (5) failure of impulse conduction in the area of the electrodes and (6) alterations in local drug or electrolyte concentrations, specifically type I antiarrhythmic agents and potassium.

While metabolic acidosis and alkalosis have been shown in dogs and in humans to result in an increase in myocardial stimulation threshold,[13-17] the import of pH changes on the intracardiac electrogram, and therefore on pacemaker sensing function, is not known. It is conceivable that metabolic acidosis, as is seen in cardiogenic shock or cardiac arrest, could cause changes in voltage or duration of intracardiac signals such that the resulting electrogram is suboptimal for sensing. Systematic study of this possibility is feasible, and warranted.

Myocardial cellular edema and ventricular dilatation have been shown to produce sufficient distortion of a depolarization signal to result in suboptimal electrograms for sensing.[6,7] Signal distortion in this setting might be due to slowing of impulse transmission and, in bipolar pacing systems, to a change in bipolar axis relative to the depolarization pathway, such that the impulse yields signals of equal magnitude at each electrode, resulting in small or zero bipolar voltage.[18] While these conditions might be expected to be associated with a concomitant increase in myocardial stimulation threshold and thus in failure to pace, their simultaneity is in fact not often observed unless the patient is moribund.

Contributing to the pacemaker-related arrhythmias in our patient could have been lower-than-normal thresholds for ventricular tachycardia and fibrillation, in turn caused by infarction, high serum concentrations

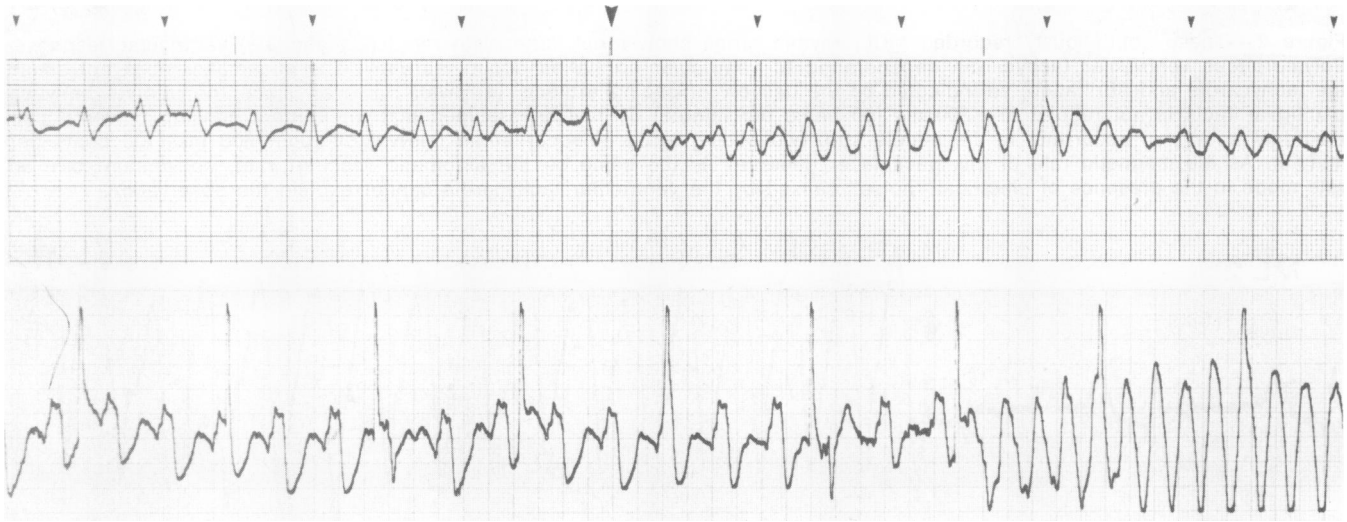

Figure 4.—These modified Lead II rhythm strips were recorded in close temporal proximity. A wide complex tachycardia (probably atrial flutter with 2:1 atrioventricular conduction) is occurring at a rate of 150 per minute. Pacemaker nonsensing is evidenced by the appearance of pacing artifacts at a regular rate of about 50 per minute **(arrows, top strip only).** In the top strip, delivery of a pacing stimulus at the end of the QRS complex **(large arrow)** results in ventricular fibrillation. In the bottom strip, a similarly timed pacing stimulus results in ventricular flutter.

of digoxin,[18-20] metabolic acidosis and probable stress-related sympathetic stimulation. In this connection it is noteworthy that infusions of epinephrine or low doses of isoproterenol and performance of physical exercise, with its attendant sympathetic stimulation, result in measurable, if transient, decreases in pacing threshold in humans.[13] The decreases in pacing threshold due to sympathetic stimulation could make a patient more vulnerable to pacemaker-induced rhythm disturbances.

Chest wall stimulation resulting in pacemaker inhibition was a lifesaving maneuver in this patient: it abolished his pacemaker-induced ventricular tachycardia and fibrillation. While use of chest wall stimulation in the setting of acute myocardial infarction with competitive rhythms has been reported,[4] its use in preventing life-threatening dysrhythmias has not been emphasized. As the technique of chest wall stimulation is easily and usually rapidly accomplished, familiarity with it by physicians managing patients with pacemakers is necessary. The prolonged use of suction cup electrodes did not pose any problems in our patient. The electrodes were secured to the chest with ample amounts of tape so as to avoid their falling off. Bleeding into the skin was not observed, and no discomfort was produced by the chest wall stimuli at the current strength used. If surface electrode stability using suction cup or paste-on skin electrodes is marginal, steel suture wires or small gauge metal needles introduced subcutaneously may be used instead and connected to the external pacemaker generator by alligator cables; these materials may be particularly useful if prolonged chest wall stimulation is anticipated.

Chest wall stimulation to inhibit implanted unipolar pacemaker generators is generally somewhat easier to accomplish than for bipolar pulse generators since in unipolar systems the pacemaker generator itself serves as the anode, resulting in wide separation between anode and cathode. In order to inhibit bipolar pacemaker generators, the surface or subcutaneous electrodes must be placed over the presumed position of two closely spaced intracardiac electrodes having an interelectrode distance of 1 to 2 cm. The surface electrodes are generally placed about 5 to 7 cm apart, to the right and left of the lower sternum, approximating the assumed location of the tip of the electrode catheter at the apex of the right ventricle. Despite the considerable time that must occasionally be devoted to achieving optimum location of the surface electrodes in patients with bipolar pacemakers, inhibition may not be achieved. Under these circumstances, consideration must be given to their removal.

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